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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/806,905	03/23/2004	David Scheinberg	D6499	2406
7590 Benjamin Aaron Adler ADLER & ASSOCIATES 8011 Candle Lane Houston, TX 77071			EXAMINER FETTEROLF, BRANDON J	
			ART UNIT 1642	PAPER NUMBER
			MAIL DATE 04/04/2008	DELIVERY MODE PAPER

**Please find below and/or attached an Office communication concerning this application or proceeding.**

The time period for reply, if any, is set in the attached communication.

### Office Action Summary

**Application No.**

10/806,905

**Applicant(s)**

SCHEINBERG ET AL.

**Examiner**

BRANDON J. FETTEROLF

**Art Unit**

1642

**Period for Reply** -- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☒ Responsive to communication(s) filed on 28 December 2007.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 1, 2, 4, 5, 8-12, 32, 49, 51-53 and 58-61 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 1-2, 4-5, 8-12, 32, 49, 51-53 and 58-61 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☐ Information Disclosure Statement(s) (PTO/SB/08)  
Paper No(s)/Mail Date \_\_\_\_\_
- 4) ☐ Interview Summary (PTO-413)  
Paper No(s)/Mail Date \_\_\_\_\_
- 5) ☐ Notice of Informal Patent Application
- 6) ☐ Other: \_\_\_\_\_

## DETAILED ACTION

### *Response to the Amendment*

The Amendment filed on 12/28/2007 in response to the previous Non-Final Office Action (8/27/2007) is acknowledged and has been entered.

Claims 1-2, 4-5, 8-12, 32, 49, 51-53 and 58-61 are currently pending and under consideration.

### **Rejections Withdrawn:**

All previous rejection are withdrawn in view of Applicants amendments. In particular, the rejections are withdrawn since the combinations used in the previous rejection did not teach or suggest administering a pharmacologically effective dose of a competitive metal blocker consisting of bismuth subnitrate or bismuth subcitrate in combination with a chelator and a diuretic.

### **New Rejections Necessitated by Amendment:**

#### *Claim Rejections - 35 USC § 103*

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

Claims 1-2, 4-5, 8, 10-11, 32, 49, 51-53 and 59-60 are rejected under 35 U.S.C. 103(a) as being unpatentable over Kennel et al. (Cancer Biotherapy & Radiopharmaceuticals 2000; 15: 235-244, *of record*) in view of Satoh et al. (Eur. J. Cancer Clin Oncol. 1989; 25: 1727-1731), Jones et al. (Nuclear Medicine & Biology 1996; 23: 105-113, *of record*), and Schilcher et al. (J. Can. Res. Clin. Oncol. 1984; 107: 57-60, *of record*) in further view of Nair et al. (J. Radiat. Res. 2001; 42: 21-37).

Kennel et al. teach a method of treating lung cancer with alpha particles comprising administering a pharmacologically effective dose <sup>225</sup>Ac bound to a HEHA-MAb 210B conjugate

(abstract). The reference further teaches that while the isotope coupled to the targeting monoclonal antibody delivers a tumoricidal dose to the lung, the radiotoxicity associated with decay daughter isotopes released from the target organ limit the effectiveness of the therapy (page 242, 2<sup>nd</sup> column, last paragraph). For example, Kennel et al. teach at necropsy, animals had total ablation of bone marrow cells, splenic atrophy, some damage to the lining of their stomachs and intestine and excess accumulation of undigested food in their stomachs (page 240, 1<sup>st</sup> column, paragraph bridging page 239).

Kennel et al. do not explicitly teach administering a competitive metal blocker such as bismuth subnitrate, a chelator such as DMPS or a diuretic such as furosemide in combination with the <sup>225</sup>Ac conjugate.

Satoh et al. teach the effects of preinduction of metallothionein (MT) by bismuth subnitrate (BSN) on the adverse effects and antitumor activity of  $\gamma$ -ray irradiation in mice (abstract). In particular, the reference teaches that oral administration of BSN markedly reduced the lethal effects and bone marrow damage by  $\gamma$ -ray irradiation without compromising the tumor-reducing effect (page 1730, 1<sup>st</sup> column, last paragraph). As such, Satoh et al. teach that bismuth subnitrate pretreatment is an effective method for protection against side-effects in radiotherapy (abstract).

Jones et al. teach that a problem with the clinical use of <sup>212</sup>Bi or <sup>212</sup>Pb RITs (radioimmunoconjugates) is the potential for radiotoxicity as a consequence of either premature release of the metal by the chelate agent or metabolic catabolism of the RIT releasing from the radiometal (page 105, 2<sup>nd</sup> column 1<sup>st</sup> full paragraph). For example, the reference teaches that previous studies have identified the kidney as being potential targets for dose limitation toxicity from radio metal deposition of bismuth radioimmunoconjugates due to the presence of heavy metal binding proteins (page 109, 2<sup>nd</sup> column, 1<sup>st</sup> paragraph and page 112, 1<sup>st</sup> column, 1<sup>st</sup> full paragraph). As a way to circumvent this potential limitation, Jones et al. disclose the evaluation of the dithiol agents, 2,3-dimercapto-1-propanesulfonic acid (DMPS) and meso-2,3-dimercaptosuccinic acid (DMSA), for their use as adjuvants to reduce or prevent radiotoxicity of Lead-212 or Bismuth-212 alpha-radioimmunoconjugates. For example, the reference teaches the administration of DMPS or DMSA to mice 48 hours prior to receiving Bismuth acetate and maintaining the administration of the chelating agents for 72 hours post injection (page 109, 2<sup>nd</sup> column, 1<sup>st</sup> paragraph). Specifically, the reference teaches that administration of DMPS accelerated body clearance of bismuth and dramatically

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reduced early and late accumulation of bismuth in the kidney (page 112, 2<sup>nd</sup> column, *Conclusion*).

Schilcher et al. teach the use of furosemide, a diuretic, for the prevention of cumulative nephrotoxicity in a phase II evaluation of fractionated low and single high dose cisplatin in various tumors (abstract).

Nair et al. teach radioprotector in radiotherapy. In particular, the reference teaches that while acute toxicity has been a main reason for radioprotectors failure in clinical applications, the use of nontoxic amounts of several radioprotectors having a different mechanism of action can overcome the problems associated with their toxicity.

Thus, it would have been obvious to one of ordinary skill in the art at the time the invention to combine the teachings of the references so as to modify the method taught by Kennel et al. to include administration of a metal blocker such as bismuth subnitrate, a chelator such as 2,3-dimercapto-1-propanesulfonic acid (DMPS) and a diuretic such as furosemide in view of the teachings of Satoh et al., Jones et al. and Schilcher et al.. One would have been motivated to do so because each of the references teach that the agents are effective at reducing toxicities associated with radiotherapies. Moreover, as taught by Nair et al., combining several radioprotectors having a different mechanism of action can overcome problems associated with radioprotector toxicity. Thus, the instant situation is amenable to the type of analysis set forth in *In re Kerkhoven*, 205 USPQ 1069 (CCPA 1980) wherein the court held that it is prima facie obvious to combine two compositions each of which is taught by the prior art to be useful for the same purpose in order to for a third composition that is to be used for the very same purpose since the idea of combining them flows logically from their having been individually taught in the prior art. Applying the same logic to the instant process claims, one of ordinary skill in the art would have a reasonable expectation of success that by modifying the method taught by Kennel et al. to include administration of a metal blocker such as bismuth subnitrate, a chelator such as 2,3-dimercapto-1-propanesulfonic acid (DMPS) and a diuretic such as furosemide in view of the teachings of Satoh et al., Jones et al. and Schilcher et al., one would achieve a method for reducing the accumulation of <sup>213</sup>Bi in the kidney, as well as bone marrow damage.

Claims 1-2, 4-5, 8-12, 32, 49, 51-53 and 58-61 are rejected under 35 U.S.C. 103(a) as being unpatentable over Scheinberg et al. (US 2002/0058007, 2002, *of record*) in view of Satoh et al. (Eur. J.

Cancer Clin Oncol. 1989; 25: 1727-1731), Jones et al. (Nuclear Medicine & Biology 1996; 23: 105-113, *of record*), and Schilcher et al. (J. Can. Res. Clin. Oncol. 1984; 107: 57-60, *of record*) and in further view of Nair et al. (J. Radiat. Res. 2001; 42: 21-37).

Scheinberg et al. teach a method of treating cancerous cells with alpha particles comprising administering a pharmacologically effective dose of an  $^{225}\text{Ac}$  conjugate comprising a functionalized chelate (page 2, paragraph 0016). With regards to the cancer, the publication teaches (page 4, paragraph 0037) that cancers include, but are not limited to, prostate cancer, lymphoma, leukemia, neuroblastoma, breast and ovarian cancer. With regards to the  $^{225}\text{Ac}$  conjugate, the publication teaches (page 2, paragraph 0017) that the conjugate consists of a monoclonal antibody covalently attached to a metal chelate that complexes with  $^{225}\text{Ac}$ , wherein internalization of  $^{225}\text{Ac}$  into the cells permits the emission of alpha particles or its daughters such as  $^{221}\text{Fr}$  and  $^{213}\text{Bi}$ . For example, Scheinberg et al. provides (page 2, paragraph 0021) an  $^{225}\text{Ac}$  conjugate consisting of  $^{225}\text{Ac}$ , HuM195 antibody and DOTA as the chelating agent. Moreover, the publication discloses the toxicity of  $^{225}\text{Ac}$  constructs, wherein histological analysis of deceased mice showed gastrointestinal mucosal sloughing and bone marrow hypoplasia, consistent with severe radiotoxicity (column 8, paragraph 0097).

Scheinberg et al. does not explicitly teach administering a competitive metal blocker such as bismuth subnitrate, a chelator such as DMPS or a diuretic such as furosemide in combination with the  $^{225}\text{Ac}$  conjugate.

Satoh et al. teach the effects of preinduction of metallothionein (MT) by bismuth subnitrate (BSN) on the adverse effects and antitumor activity of  $\gamma$ -ray irradiation in mice (abstract). In particular, the reference teaches that oral administration of BSN markedly reduced the lethal effects and bone marrow damage by  $\gamma$ -ray irradiation without compromising the tumor-reducing effect (page 1730, 1<sup>st</sup> column, last paragraph). As such, Satoh et al. teach that bismuth subnitrate pretreatment is an effective method for protection against side-effects in radiotherapy (abstract).

Jones et al. teach that a problem with the clinical use of  $^{212}\text{Bi}$  or  $^{212}\text{Pb}$  RITs (radioimmunoconjugates) is the potential for radiotoxicity as a consequence of either premature release of the metal by the chelate agent or metabolic catabolism of the RIT releasing from the radiometal (page 105, 2<sup>nd</sup> column 1<sup>st</sup> full paragraph). For example, the reference teaches that previous studies have identified the kidney as being potential targets for dose limitation toxicity from

radio metal deposition of bismuth radioimmunoconjugates due to the presence of heavy metal binding proteins (page 109, 2<sup>nd</sup> column, 1<sup>st</sup> paragraph and page 112, 1<sup>st</sup> column, 1<sup>st</sup> full paragraph). As a way to circumvent this potential limitation, Jones et al. disclose the evaluation of the dithiol agents, 2,3-dimercapto-1-propanesulfonic acid (DMPS) and meso-2,3-dimercaptosuccinic acid (DMSA), for their use as adjuvants to reduce or prevent radiotoxicity of Lead-212 or Bismuth-212 alpha-radioimmunotherapy. For example, the reference teaches the administration of DMPS or DMSA to mice 48 hours prior to receiving Bismuth acetate and maintaining the administration of the chelating agents for 72 hours post injection (page 109, 2<sup>nd</sup> column, 1<sup>st</sup> paragraph). Specifically, the reference teaches that administration of DMPS accelerated body clearance of bismuth and dramatically reduced early and late accumulation of bismuth in the kidney (page 112, 2<sup>nd</sup> column, *Conclusion*).

Schilcher et al. teach the use of furosemide, a diuretic, for the prevention of cumulative nephrotoxicity in a phase II evaluation of fractionated low and single high dose cisplatin in various tumors (abstract).

Nair et al. teach radioprotector in radiotherapy. In particular, the reference teaches that while acute toxicity has been a main reason for radioprotectors failure in clinical applications, the use of nontoxic amounts of several radioprotectors having a different mechanism of action can overcome the problems associated with their toxicity.

Thus, it would have been obvious to one of ordinary skill in the art at the time the invention to combine the teachings of the references so as to modify the method taught by Kennel et al. to include administration of a metal blocker such as bismuth subnitrate, a chelator such as 2,3-dimercapto-1-propanesulfonic acid (DMPS) and a diuretic such as furosemide in view of the teachings of Satoh et al., Jones et al. and Schilcher et al.. One would have been motivated to do so because each of the references teach that the agents are effective at reducing toxicities associated with radiotherapies. Moreover, as taught by Nair et al., combining several radioprotectors having a different mechanism of action can overcome problems associated with radioprotector toxicity. Thus, the instant situation is amenable to the type of analysis set forth in *In re Kerkhoven*, 205 USPQ 1069 (CCPA 1980) wherein the court held that it is prima facie obvious to combine two compositions each of which is taught by the prior art to be useful for the same purpose in order to for a third composition that is to be used for the very same purpose since the idea of combining them flows logically from their having been individually taught in the prior art. Applying the same

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logic to the instant process claims, one of ordinary skill in the art would have a reasonable expectation of success that by modifying the method taught by Kennel et al. to include administration of a metal blocker such as bismuth subnitrate, a chelator such as 2,3-dimercapto-1-propanesulfonic acid (DMPS) and a diuretic such as furosemide in view of the teachings of Satoh et al., Jones et al. and Schilcher et al., one would achieve a method for reducing the accumulation of  $^{213}\text{Bi}$  in the kidney, as well as bone marrow damage.

Claims 1-2, 4-5, 8-12, 32, 49, 51-53 and 58-61 are rejected under 35 U.S.C. 103(a) as being unpatentable over McDevitt et al. (Science 2001; 294: 1537-1540, *of record*) in view of Satoh et al. (Eur. J. Cancer Clin Oncol. 1989; 25: 1727-1731), Jones et al. (Nuclear Medicine & Biology 1996; 23: 105-113, *of record*), and Schilcher et al. (J. Can. Res. Clin. Oncol. 1984; 107: 57-60, *of record*) and in further view of Nair et al. (J. Radiat. Res. 2001; 42: 21-37).

McDevitt et al. teach a method of treating cancerous cells with alpha particles comprising administering a pharmacologically effective dose of an  $^{225}\text{Ac}$  conjugate comprising a functionalized chelate (page 1537, Abstract). With regards to the cancer, the reference teaches (page 1537, Abstract) that cancers include, but are not limited to, prostate cancer, lymphoma, leukemia, neuroblastoma, breast and ovarian cancer. With regards to the  $^{225}\text{Ac}$  conjugate, the reference teaches (page 1538, 1<sup>st</sup> column, 2<sup>nd</sup> full paragraph) that the conjugate consists of a monoclonal antibody covalently attached to a metal chelate that complexes with  $^{225}\text{Ac}$ , wherein internalization of  $^{225}\text{Ac}$  into the cells permits the emission of alpha particles or its daughters such as  $^{221}\text{Fr}$  and  $^{213}\text{Bi}$ . For example, Scheinberg et al. provides (page 1538, 1<sup>st</sup> column, 2<sup>nd</sup> full paragraph) an  $^{225}\text{Ac}$  conjugate consisting of  $^{225}\text{Ac}$ , HuM195 antibody and DOTA as the chelating agent. Moreover, the publication discloses the biodistribution of  $^{225}\text{Ac}$  conjugates in tumor bearing mice, wherein the results demonstrated specific tumor uptake of  $^{225}\text{Ac}$ , but  $^{213}\text{Bi}$ , e.g. *daughter of  $^{225}\text{Ac}$* , accumulation in the kidney as a result of decay of the daughters from nontargeted constructs (page 1538, Figure 1B).

McDevitt et al. does not explicitly teach administering a diuretic such as furosemide, a dithiol chelate and a metal blocker such as bismuth subnitrate in combination with the  $^{225}\text{Ac}$  conjugate.

Satoh et al. teach the effects of preinduction of metallothionein (MT) by bismuth subnitrate (BSN) on the adverse effects and antitumor activity of  $\gamma$ -ray irradiation in mice (abstract). In



particular, the reference teaches that oral administration of BSN markedly reduced the lethal effects and bone marrow damage by  $\gamma$ -ray irradiation without compromising the tumor-reducing effect (page 1730, 1<sup>st</sup> column, last paragraph). As such, Satoh et al. teach that bismuth subnitrate pretreatment is an effective method for protection against side-effects in radiotherapy (abstract).

Jones et al. teach that a problem with the clinical use of  $^{212}\text{Bi}$  or  $^{212}\text{Pb}$  RICs (radioimmunoconjugates) is the potential for radiotoxicity as a consequence of either premature release of the metal by the chelate agent or metabolic catabolism of the RIT releasing from the radiometal (page 105, 2<sup>nd</sup> column 1<sup>st</sup> full paragraph). For example, the reference teaches that previous studies have identified the kidney as being potential targets for dose limitation toxicity from radio metal deposition of bismuth radioimmunoconjugates due to the presence of heavy metal binding proteins (page 109, 2<sup>nd</sup> column, 1<sup>st</sup> paragraph and page 112, 1<sup>st</sup> column, 1<sup>st</sup> full paragraph). As a way to circumvent this potential limitation, Jones et al. disclose the evaluation of the dithiol agents, 2,3-dimercapto-1-propanesulfonic acid (DMPS) and meso-2,3-dimercaptosuccinic acid (DMSA), for their use as adjuvants to reduce or prevent radiotoxicity of Lead-212 or Bismuth-212 alpha-radioimmunotherapy. For example, the reference teaches the administration of DMPS or DMSA to mice 48 hours prior to receiving Bismuth acetate and maintaining the administration of the chelating agents for 72 hours post injection (page 109, 2<sup>nd</sup> column, 1<sup>st</sup> paragraph). Specifically, the reference teaches that administration of DMPS accelerated body clearance of bismuth and dramatically reduced early and late accumulation of bismuth in the kidney (page 112, 2<sup>nd</sup> column, *Conclusion*).

Schilcher et al. teach the use of furosemide, a diuretic, for the prevention of cumulative nephrotoxicity in a phase II evaluation of fractionated low and single high dose cisplatin in various tumors (abstract).

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Thus, it would have been obvious to one of ordinary skill in the art at the time the invention to combine the teachings of the references so as to modify the method taught by McDewitt to include administration of a metal blocker such as bismuth subnitrate, a chelator such as 2,3-dimercapto-1-propanesulfonic acid (DMPS) and a diuretic such as furosemide in view of the

teachings of Satoh et al., Jones et al. and Schilcher et al.. One would have been motivated to do so because each of the references teach that the agents are effective at reducing toxicities associated with radiotherapies. Moreover, as taught by Nair et al., combining several radioprotectors having a different mechanism of action can overcome problems associated with radioprotector toxicity. Thus, the instant situation is amenable to the type of analysis set forth in In re Kerkhoven, 205 USPQ 1069 (CCPA 1980) wherein the court held that it is prima facie obvious to combine two compositions each of which is taught by the prior art to be useful for the same purpose in order to form a third composition that is to be used for the very same purpose since the idea of combining them flows logically from their having been individually taught in the prior art. Applying the same logic to the instant process claims, one of ordinary skill in the art would have a reasonable expectation of success that by modifying the method taught by McDevitt to include administration of a metal blocker such as bismuth subnitrate, a chelator such as 2,3-dimercapto-1-propanesulfonic acid (DMPS) and a diuretic such as furosemide in view of the teachings of Satoh et al., Jones et al. and Schilcher et al., one would achieve a method for reducing the accumulation of  $^{213}\text{Bi}$  in the kidney, as well as bone marrow damage.

Therefore, No Claim is allowed.

### ***Conclusion***

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to BRANDON J. FETTEROLF whose telephone number is (571)272-2919. The examiner can normally be reached on Monday through Friday from 7:30 to 4:30.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Larry Helms can be reached on 571-272-0832. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Brandon J Fetterolf, PhD  
Primary Examiner  
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/Brandon J Fetterolf, PhD/  
Primary Examiner, Art Unit 1642